HERITABILITY OF RESISTANCE TO *Cronartium fusiforme* IN *Pinus elliottii* AS AFFECTED BY DISEASE INCIDENCE AND A COMPARISON OF BREEDING PROCEDURES

By

SUNG IN SOHN

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HERITABILITY OF RESISTANCE TO *Cronartium fusiforme* IN *Pinus elliottii* AS AFFECTED BY DISEASE INCIDENCE AND A COMPARISON OF BREEDING PROCEDURES

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Sung In Sohn

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A total of 143 open-pollinated families from a slash pine population selected for growth traits but unselected for rust (population-A) and 92 open-pollinated families from a slash pine population selected for fusiform rust resistance (population-B) were planted in three high rust hazard locations: Taylor County, Florida, and Webster and Bulloch Counties, Georgia, over a two-year period (1971-1972). Each family was represented in a ten-tree row with three replications in each location. Fusiform rust infection data were assessed in three different forms: binomial (1 or 0), plot mean (percentage), and angular transformed plot mean data.

Results showed that substantial variation in rust resistance existed among families in the selected population as well as in the unselected population. There was an indication of location x family interaction in the 1971 plantings, especially due to the Taylor County, Florida, site, but the

magnitude of the interaction was small. Location x family interaction was not statistically significant in the 1972 plantings.

Relationships between estimates of heritability and infection percentage of each test were somewhat different in two forms of data: binomial and transformed plot mean data. Estimates of heritability increased linearly as infection percentage increased in binomial data, while estimates of heritability in transformed plot mean data increased up to 60% infection level and decreased at higher infection levels. With adjustment, the estimate of heritability of rust resistance of individual trees was constant ($h^2 = 0.281$) over infection levels ranging from 40 to 85%.

Predicted and realized gains from mass selection, mass selection plus progeny testing, and family and within family selection were computed for both populations. High infection level (high selection pressure) was the most important factor for maximization of genetic gain from mass selection (20 percentage points), although maximum gain was expressed from tests averaging 71.0% infection. Expected gain from mass selection estimated with binomial data was much closer to realized gain than with transformed plot mean data. Mass selection plus progeny testing was the most efficient system (approximately 23 and 16 percentage points for population-A and B, when the best 25% of trees were selected) followed by family and within family selection (approximately 18 and 12 percentage points). Establishing seed orchards through

recurrent rust-free selection in high rust hazard areas might be the best alternative to meet the challenge of pathogenic variability in the fungus while maintaining moderate gain (approximately 10% and 6%).

Selection in test sites where genotype x environment interaction was suspected and infection level was very low resulted in no improvement in resistance.

INTRODUCTION

Rapid increase of fusiform rust, caused by Cronartium fusiforme Hedge. and Hunt ex Cumm., in Southern United States became the most serious problem in establishing slash pine (Pinus elliottii Engelm var. elliottii) and loblolly pine (P. taeda L.) plantations. The increase in young pine plantations and in disease incidence during the last two decades has developed an epidemic situation in much of this region. In Florida and Georgia, the rust incidence in slash pine plantations increased at a rate of 2-3% per year from 1950 to 1967 (Schmidt et al., 1974). A Southwide survey for rust incidence indicated a stumpage loss caused by fusiform rust of $28 million in 1972 (Powers et al., 1974).

Chemical fungicides, which may be appropriate for rust control in forest nurseries, are not feasible to apply in forests generally because of economic and ecological restrictions.

One of the alternatives, genetic control, utilizing substantial variation in resistance to fusiform rust existing in slash and loblolly pines could be the most promising mode of defense and of major economic value for southern forest industries. The efficient utilization of this control measure depends on (1) the degree of additive genetic control

of resistance; (2) the stability of resistance over a range of environments, time as well as space-wise; and (3) the selection of proper breeding programs.

Estimates of heritability provide useful information for planning breeding procedures for genetic improvement. However, there are difficulties of estimating heritability of rust resistance as it can be considered a threshold, all-or-none characteristic. The conventional way to handle data of this type is to use angular transformed plot mean data which tends to provide a normal distribution. By this procedure good estimates of family mean heritability can be calculated and heritability for individual trees can be also provided indirectly using constant binomial sampling variance (Becker and Marsden, 1972).

However, it is difficult to interpret the results of genetic gain calculation based on heritabilities after transformation. Heritability for individual trees can be estimated with no difficulty when each tree is numerically evaluated. For instance, a severity index or a count of galls per tree which tend to be normally distributed can be easily handled (Blair, 1970; Barker, 1973). But a shortcoming of this method is interpreting given unit of gain from selection. In this study, estimates of genetic parameters of rust resistance computed from binomial data and from transformed plot mean data are compared. The binomial approach, in particular, permits estimates of genetic parameters of rust resistance considered as a

threshold character and their use in predicting genetic gain.

The objectives of this study are;

(1) To estimate the amount of variability in resistance to fusiform rust.

(2) To compare estimates of genetic parameters computed from three different approaches to data analysis, binomial (p-scale) data, plot mean (percentage) data, and angular transformed plot mean data.

(3) To define the relationships between the average infection levels and genetic parameters of several test populations.

(4) To find an optimum infection level for maximization of genetic gain.

(5) To predict the gain in resistance that can be expected from several methods of selection commonly used in tree breeding programs.

LITERATURE REVIEW

In early references, from Biblical times to the late 19th century, the existence of various species of rust attacking cereals has been reported. The earliest mention is in the Bible (Deut. 28:22), which would place the date about 1360 B.C. The Hebrew term "yerageon" (yellowing) has been rendered "mildew" in English. Later, mildew, rust, and blight were used interchangably (Arthur, 1929).

Recent severe damage of wheat and corn crops by stem rust and corn blight, among the most striking epidemic situations in annual crops, stimulated a massive volume of literature concerning the problem (National Academy of Sciences, 1972; Ullstrup, 1972). Even though forest tree diseases are of a somewhat different nature, the annual crop breeding programs suggest possible resistance breeding procedures.

Cronartium fusiforme, which causes fusiform rust in southern pines, seems to have existed for a long time in the southern part of the United States (Duffield, 1972). The earlier reports dated in 1929 (Hays and Wakely, cited in Czabator, 1971) described C. fusiforme as a rare disease of minor importance. Increase of fusiform rust has been very rapid since then. By 1947, Siggers and Lindgren had reported

a plantation infected over 90% in southern Mississippi. A thorough investigation of the fusiform rust incidence over the southern part of the United States through the Fusiform Rust Incidence Survey has been completed indicating widespread incidence of the disease (Phelps, 1974; Schmidt et al., 1974; Walterscheidt and Van Arsdel, 1976). At the same time, damage due to this disease, estimated originally at 281 million board feet of pine saw timber and 97 million cubic feet of growing stock (U.S.D.A., 1958), was updated in 1974 by doubling these figures (Phelps and Chellman, 1974; Powers et al., 1974). The annual stumpage value loss of 20 million dollars also was raised to 28 to 30 million. Such a rapid increase may be due to upsetting the natural balance by off-site or out-of-range planting (Czabator, 1971; Dinus, 1974).

Attempts to eradicate the susceptible oaks within 1500 feet of plantations were not successful in controlling fusiform rust (Waggoner, 1962). Fertilization and site preparation had a negative effect on fusiform rust control in southern pines (Dinus and Schmidtling, 1971; Miller, 1972; Blair and Cowling, 1974; Hollis et al., 1975). Southern pine nurseries are routinely sprayed with ferbam during the period of natural spore cast, normally with very satisfactory control of fusiform rust. Soil application of systemic fungicides seems to be effective in controlling the fusiform rust in nurseries (Hare, 1973), though negative results have been reported (Rowan, 1972). Breeding for resistance has been proven to be very promising and may be the only

feasible way to control many forest diseases. The basis for breeding work lies in variation existing among trees and their ability to transfer the trait to next generation. Sources of resistance can be found through analyses of variation, and the maximal utilization of these sources of resistance will determine the efficiency of the breeding work.

Variation

Slash and loblolly pines are the most highly susceptible among southern pine species. However, slash pine usually has the most severe damage and mortality (Jewell, 1960, Czabator, 1971; Powers, 1975). Shortleaf pine is considered essentially immune to fusiform rust and its hybrids are highly resistant (Henry and Bercaw, 1956; Jewell, 1959; Schmitt, 1968), even though all of these hybrids were infected through artificial inoculation (Jewell, 1961), whereas longleaf pine and its hybrids showed only moderate resistance (Derr, 1966). Progenies from loblolly x slash crosses, on the other hand, were much more susceptible than any other hybrids or their parental species (Schmitt, 1968).

Several progeny tests have shown that substantial variation in resistance to rust exists for slash (Barber, 1961, 1964; Schmidt and Goddard, 1971) and loblolly pines (Barber, 1966; Kinloch, 1968; Kinloch and Stonecypher, 1969; Kinloch and Zoerb, 1971). Resistance and susceptibility is under strong genetic control and the mode of the inheritance

is probably polygenic (Jewell and Mallett, 1967; Schmitt, 1968). Much of this information resulted from artificial inoculation experiments in both species (Goddard and Arnold, 1966; Goddard and Schmidt, 1971; Jewell and Mallett, 1964, 1967; Kinloch and Kelman, 1965).

The southwide seed source study (Wells and Wakeley, 1966) showed a consistent and distinctive geographic variation in infection in loblolly pine. In every case, the most resistant sources were from near the extremities of the species range: west of the Mississippi River; Livingston Parish, Louisiana; Eastern Maryland; and Marion County, Florida (Wells and Switzer, 1971; Draper, 1975).

In contrast, no clear geographic pattern was found in slash pine (Snyder et al., 1967; Gansel et al., 1971). A higher frequency of resistant clones seems to be from highly infected areas in the center of the natural range, which is in direct contrast to the situation in loblolly pine, where more resistance was found in trees from the extremities of the natural range, where little rust occurs (Sohn et al., 1975).

There are some speculations on the differences between rust susceptibility patterns in these two closely related species. Wells and Wakeley (1966) proposed that varying degrees of selection pressure imposed by the rust in different parts of its host range, and long association with the alternate host and presumably with rust (Wahlenberg, 1960), led to co-evolution of both sides, pine and rust.

Greater tolerance has been developed in the center part of the loblolly population but highly resistant materials were produced at the extremities of their range. Slash pine, however, was originally restricted over much of its range to pond margins and had very little association with alternate hosts. Widespread planting of slash pine in other habitats was equivalent to exposing the species to a recently introduced disease (Goddard and Wells, 1976).

An alternative but plausible explanation for the distinctive geographic pattern in loblolly pine seems to be introgression of shortleaf pine genes into loblolly pine (Wells and Wakeley, 1966; Kinloch and Stonecypher, 1969; Kinloch, 1972). Hare and Switzer (1969) found biochemical evidence to support this theory. But this theory does not explain the source of resistance in Marion County, Florida.

Discrepancy between these two species also can be found in genotype x environment interaction. Strong genotype x environment interactions between seed sources and planting site (Gansel et al., 1971), and relatively large site x progeny and year x progeny interactions (Rockwood and Goddard, 1973; Sohn et al., 1975) were also observed in slash pine. But in general, genotype x environment interaction in growth traits is larger in slash pine than the moderate genotype x environment interaction in resistance to fusiform rust. In contrast, genotype x environment interaction was not significant in several studies of loblolly pine (Kraus, 1967; Kinloch, 1968; Blair, 1970). It is well

understood that the stability of genotypes in several environments is an important factor for successful breeding work. For rust resistance in particular, two hosts and a parasite are involved as well as environmental factors. Fungi have numerous opportunities for natural mutation and testing of resistance in trees repeated over time and space may well be necessary.

Methods of Testing Rust Resistance

Artificial inoculation has been extensively used to detect susceptible trees at an early stage since the technique was devised (Jewell, 1960). Methods of artificial inoculation were well documented by Schmidt (1972). More recent modified artificial inoculation methods were devised to meet the precision and efficiency this technique requires (Powers, 1971; Snow and Kais, 1972; Matthews and Rowan, 1972; Hare, 1973; Dinus and Hare, 1974; Laird and Phelps, 1975).

Artificial inoculation using different sources of inoculum confirmed the existence of pathogenic variability in C. fusiforme (Snow et al., 1969, 1972, 1975; Snow and Kais, 1970). Pathogenic variability was very high and variation was as large between galls in the same trees as between locations.

Correlations between artificial inoculation and field tests were good in several studies (Dinus, 1969; Wells and Dinus, 1974) suggesting that artificial inoculation is a very efficient method to predict field performance, while

some showed discrepancy (Sohn, 1975; Goddard and Schmidt, 1971). Field tests are very important to determine accurate genetic parameters in rust resistance especially when the relationship between artificial inoculation and field tests are poor. Goddard and Schmidt (1971) artificially inoculated progenies of selected rust-free trees from a Georgia plantation having 90% rust incidence. The result was rather confusing because the selected lots were as heavily infected as check lots. They pointed out the possibility of variation in rust fungus as one of the reasons for their conflicting findings. It is very important to include many sources of inoculum in artificial inoculation to predict performance of trees in fields, since the pathogenic variability in many areas has been proven.

Rust Resistance Mechanisms

Several general types of resistance mechanisms have been reported in southern pines. Histological examination of inoculated slash pine seedlings by Miller et al. (1976) provided four general types of host responses. The four types are (1) no apparent penetration or infection, (2) subliminal infection, (3) hypersensitive reactions, and (4) normal gall development. They identified a type (1) as a potential and type (3) as a definite form of resistance. Even a definite form of resistance, type (3), had three general types of reaction zones indicating several factors

involved in resistance. Mechanisms of rust resistance may be due to (1) morphological and anatomical barriers (Jewell, 1960; Powers, 1968; Jewell and Snow, 1972), and (2) physiological and biochemical substances (Hare, 1970, 1972; Rockwood, 1973; Lewis, 1973). Nothing is known about the inheritance pattern of these different mechanisms.

Estimates of Heritability

Published estimates of heritability for rust resistance are highly variable. Estimates in slash pine varied from one planting (0.054) to another (0.238) in progeny tests (Rockwood and Goddard, 1973). Goddard and Arnold (1966) reported 0.199 as an estimate from artificial inoculation. Although the estimates for individual trees were low and extremely variable, estimates of progeny means were high, often exceeding 0.6 (Rockwood and Goddard, 1973).

Estimates tended to increase with rising infection rate. Correlations among heritabilities of individuals and infection rates were about 0.40 and the positive trend was evident up to the maximum infection rate in the study (70%) (Rockwood and Goddard, 1973). Chances of "escape" at low levels of infection are reasonably understood. Minimum and maximum infection rates of tests to yield the optimum genetic parameters have never been determined. The Florida Cooperative Forest Genetics Research Program uses a weighting system where each test is weighted linearly according to the infection level. This linear relationship was selected

arbitrarily rather than from published evidence. Kinloch and Kelman (1965) contend that severe artificial inoculation (very high average infection rate) may not be a valid index.

In loblolly pine, the heritability estimates for the number of galls per tree, severity index, and percentage of trees infected were 0.29, 0.22, and 0.20 in one planting and 0.09, 0.04, and 0.12 in another planting (Blair, 1970). Family heritabilities over four different environments were consistently high (0.65 to 0.85) in the study by Kinloch and Stonecypher (1969).

Estimates of heritability can vary according to the way in which infection is tabulated. Percentage values expressed as the number of trees infected per plot or the angular transformation of the percentages have primarily been used for rust data (Becker and Marsden, 1972; Rockwood and Goddard, 1973).

Estimation of heritability for individual trees using a method suggested by Robertson and Lerner (1949) has been used often in rust data (Blair, 1970; Goddard and Arnold, 1966). Estimates from this method also represent heritability of binomial scale (p-scale) but it can be a biased estimate depending on the non-additive portion included in computation.

Rust infection behaves as a typical threshold character, and should be amenable to analysis as a binomially distributed character, though there seem to be no reports in the literature where this has been done. Theoretically (Robertson and Lerner, 1949), the heritability estimate on

binomial scale is $h^2_b = h^2 z^2/p(1 - p)$, where h^2 is heritability on normal scale and z is the height of the ordinate of the normal distribution at the threshold point corresponding to a fraction p of the population having the character. Later, other researchers (Dempster and Lerner, 1950; Van Vleck, 1972) compared these two heritability values in various circumstances and concluded that estimates from p-scale can be used correctly when heritability is low and the incidence is not too close to zero or unity.

Genetic Gain and Breeding Methods

Phenotypic selection from heavily infected areas seems to be a very effective breeding method in slash pine (Goddard et al., 1975). Progenies of rust-free selections in heavily infected plantations were infected with an average disease percentage of 61.2%, whereas progenies from clones of low selection pressure on rust resistance showed an average of 85.6% infection. Dinus (1969) also reported a similar situation with a limited population size in slash pine. Selection of uninfected parent trees from natural stands yielded an important initial gain of approximately 15% of the mean in loblolly pine (Blair, 1970). Similar results also were reported in the study by Kinloch and Stonecypher (1969). Phenotypic selection resulted in progeny with an average incidence of rust 19.3% less than that of bulk seedlings after artificial inoculation (Dinus, 1970).

Several others (Jewell and Mallett, 1967; Kinloch and Kelman, 1965) reported similar results.

Selection based on subsequent progeny testing yielded an additional gain of a magnitude similar to mass selection in loblolly pine (Blair, 1970).

Artificial inoculation methods seem potentially to be useful for rust resistance breeding in southern pines (Dinus and Griggs, 1975). When rust-free individuals following artificial inoculation were planted in the field with non-inoculated control trees, rust-free individuals had ten times fewer galls per tree than non-inoculated controls. The average infection rate was 2% for rust-free and 17% for control trees. Similar results were reported by Powers et al. (1976). After artificial inoculation, the average rust infection for half-sib families ranged from 25 to 51% in contrast to 78% for commercial check. When the healthy survivors were outplanted, the frequency of infection after two years in the field was 3 to 7% for resistant families and 26% for the checks.

Roguing established clonal orchards or establishing reconstituted orchards after the progeny testing can be a very effective program for short term gain. Brunswick Paper and Pulp Company is establishing a new orchard including their top 31% resistant clones for an improvement of as much as 28% over commercial checks (Rockwood and Goddard, 1973).

MATERIALS AND METHODS

General information on the high hazard tests and slash pine clones included in this study is outlined in several published papers (Goddard et al., 1975; Sohn et al., 1975), and especially in the Report of the Florida Cooperative Forest Genetics Research Program (Goddard et al., 1974). Therefore, only the information necessary to interpret the data will be given in this paper.

Populations

Two different types of populations were used. A population of slash pine clones, selected for growth (volume) in the Florida Cooperative Forest Genetics Research Program was considered to be a wild population in regard to resistance to fusiform rust. Several studies indicated that selection of southern pines for growth traits did not affect rust susceptibility (Barber, 1964; Woessner, 1965; Sluder, 1975).

The second population consisted of trees which were selected for rust resistance in a plantation where over 90% of the trees were infected. All infected trees were removed and seeds were collected from the remaining rust-free trees. For convenience, the first population, unselected for resistance population will be called population-A and

the second, or selected population, will be called population-B.

Planting Sites

Open-pollinated progenies of more than 300 slash pine clones from population-A were planted in three high hazard test sites which were selected on the basis of high fusiform rust incidence in nearby pine plantations. These three high hazard sites are located in Taylor County, Florida; Webster County, Georgia; and Bulloch County, Georgia. Starting in 1971, the plan was to plant progenies of all clones in the Florida Cooperative Forest Genetics Research Program at each location at least one time. Annual plantings have been continued at these locations. Only a few families were planted more than one year at a location. Progenies from population-B were also included at the same sites but only at the 1971 Webster County planting and the 1972 Taylor County and Bulloch County plantings contained enough progenies to be included in the statistical analyses.

Seedlings from both populations were planted randomly in ten-tree row plots with three blocks in each location. Spacing was 0.6 m within rows 2.4 m apart. The 1972 plantings were adjacent to but not randomized with the 1971 plantings.

Data Assessment

Rust infection was assessed in January, 1974, and January, 1975, three years after planting in each case. Fifth year rust infection data were added in January, 1976, for the 1971 plantings and in December, 1976, for the 1972 Taylor County and Bulloch County plantings. Data were recorded for individual trees as "1" if the tree showed any infection symptoms and as "0" if the tree was not infected.

Analyses

A total of 92 families from population-A were in common in all three locations in the 1971 plantings. Another group of 64 families was also planted at all three locations in the 1972 plantings. Among these families, 13 occurred in all three locations and in both planting years. This provided a total of 143 families for statistical analyses from population-A. In a similar way, 92 families were available from population-B.

Data on these families were used for three types of statistical analyses:

1. Individual tree data of "1"s and "0"s, i.e., binomial data.
2. The percentage of infected trees per plot, i.e., plot mean data.
3. Arcsin square root transformed data of the percentage of infected trees after the adjustment of

proportions to minimize fluctuation (Bartlett, 1947), i.e., transformed plot mean data.

The analysis of variance to determine the mean squares necessary for estimating components of variance is given in Table 1 for binomial data and in Table 2 for the transformed plot mean data. Least squares analyses were used to correct for missing data. The possibility of combining the two planting years was excluded because only 13 families were replicated in both years and this would create an extremely unbalanced design. Therefore, data from the two planting years were analyzed separately, providing different estimates of variance components. Data were analyzed by individual location and combined over the three locations. Expected mean squares for separate location were the same as indicated in Tables 1 and 2 with the elimination of the effect of location and interaction with location. Variance components were partitioned in population-A and used for subsequent computation. Since population-B was selected for the trait it was inappropriate for estimation of genetic parameters.

Estimates of Heritability

Individual Tree Heritability (h^2_i)

(1) From binomial data:

$$h^2_i = \frac{4\sigma^2_f}{\sigma^2_T}$$

Table 1. Analysis of variance for estimating components of variance for the resistance to fusiform rust in slash pine as a threshold character (binomial data).

Source of variation	d.f.	Expectation of mean squares
Locations*	$(\ell-1)$	$\sigma^2_t + k_{11}\sigma^2_{b(\ell)f} + k_{21}\sigma^2_{\ell f} + k_{41}\sigma^2_{b(\ell)} + k_{51}\theta^2_L$
Blocks (locations)	$\ell(b-1)$	$\sigma^2_t + k_{12}\sigma^2_{b(\ell)f} + k_{22}\sigma^2_{\ell f} + k_{42}\sigma^2_{b(\ell)}$
Families	$f-1$	$\sigma^2_t + k_{13}\sigma^2_{b(\ell)f} + k_{31}\sigma^2_f$
Locations x families	$(\ell-1)(f-1)$	$\sigma^2_t + k_{14}\sigma^2_{b(\ell)f} + k_{23}\sigma^2_{\ell f}$
Blocks (locations) x families	$\ell(b-1)(f-1)$	$\sigma^2_t + k_{15}\sigma^2_{b(\ell)f}$
Within plots	$\ell\ b\ f\ (k-1)$	σ^2_t

*All effects are random except the effect of the locations.

ℓ, b, f, k = Number of locations, blocks, families, and trees per plot

σ^2_t = Within plot variance

$\sigma^2_{b(\ell)f}$ = Variance component due to the interaction of blocks within locations and families

$\sigma^2_{\ell f}$ = Variance component due to the interaction of locations and families

σ^2_f = Variance component due to families

Table 1. (Continued)

$\sigma^2_{b(\ell)}$ = Variance component due to blocks in locations

$$h^2_i = \frac{4\sigma^2_f}{\sigma^2_t + \sigma^2_{b(\ell)f} + \sigma^2_{\ell f} + \sigma^2_f} = \frac{4\sigma^2_f}{\sigma^2_T}$$

$$h^2_f = \frac{\sigma^2_f}{\frac{\sigma^2_t}{\ell \cdot b \cdot k} + \frac{\sigma^2_{b(\ell)f}}{\ell \cdot b} + \frac{\sigma^2_{\ell f}}{\ell} + \sigma^2_f} = \frac{\sigma^2_f}{\sigma^2_{T'}}$$

Coefficients for the variance components were determined using SAS VARCOMP procedure. They were as follows:

	k_{11}	k_{12}	k_{13}	k_{14}	k_{15}	k_{21}	k_{22}	k_{23}	k_{31}	k_{41}	k_{42}	k_{51}
1971	8.99	8.96	8.95	8.76	8.44	27.61	25.17	24.75	74.23	761.37	759.48	2277
1972	9.25	9.24	9.21	9.13	8.98	27.14	27.42	26.55	80.08	570.06	569.42	1708

Table 2. Analysis of variance for the rust resistance data transformed to arcsin of the square root of the plot mean values.

Source of variation	d.f.	Expectation of mean squares
Locations	ℓ-1	
Blocks (locations)	$\ell(b-1)$	
Families	f-1	$\sigma^2_R + \ell b \sigma^2_f$
Locations x families	$(\ell-1)(f-1)$	$\sigma^2_R + b\sigma^2_{\ell f}$
Residual	$\ell(f-1)(b-1)$	$\sigma^2_R = \frac{1}{k}\sigma^2_b + \sigma^2_e$
Binomial*		$\frac{1}{k}\sigma^2_b$

ℓ, b, f, k = Number of locations, blocks, families, and trees per plot

σ^2_R = Variance due to residual effects including the interaction of blocks (locations) x families and within plot and binomial sampling error

*σ^2_b = Variance due to binomial sampling (constant)

σ^2_e = Variance due to plot

$\sigma^2_{\ell f}$ = Variance due to the interaction of locations and families

σ^2_f = Variance due to families

$$h^2_i = \frac{4\,\sigma^2_f}{\sigma^2_b + \sigma^2_e + \sigma^2_{\ell f} + \sigma^2_f} = \frac{4\,\sigma^2_f}{\sigma^2_T}$$

$$h^2_f = \frac{\sigma^2_f}{\frac{\sigma^2_R}{\ell \cdot b} + \frac{\sigma^2_{\ell f}}{\ell} + \sigma^2_f} = \frac{\sigma^2_f}{\sigma^2_{T'}}$$

where, h^2_i = estimate of heritability of individual tree value,

σ^2_f = estimate of variance component for family (quarter of additive variance),

σ^2_T = estimate of the total phenotypic variance of individual trees.

(2) From transformed plot mean data: basically the same formula was used except binomial sampling variance was substituted for within plot variance. Binomial sampling variance is merely: $\sigma^2_b = p(1 - p)$, where p is the proportion of infected trees divided by total number of trees in the plot. With the adjusted proportions transformed to arcsins, the binomial sampling variance is constant and equal to 821 (Fisher and Yates, 1948).

(3) From the method suggested by Robertson and Lerner (1949):

$$h^2 = \frac{\chi^2 - (f - 1)}{rNo}$$

where, f = number of families

r = genetic relationship within families

$$No = \sum n_i - \frac{\sum n_i^2}{\sum n_i} - (f - 1)$$

(n_i = number of individuals in the i^{th} family)

χ^2 = the heterogeneity Chi-square (see example in Goddard and Arnold, 1966).

Family Heritability (h^2_f)

Percentage data and transformed data of plot means can be used to estimate family mean heritability, as

$$h^2_f = \frac{\sigma^2_f}{\sigma^2_{T'}}$$

where,

h^2_f = estimate of heritability based on family mean

$\sigma^2_{T'}$ = estimate of the total phenotypic variance of plot means.

Family heritability can also be estimated from binomial data. Estimates of the total phenotypic variance of plot means ($\sigma^2_{T'}$) were calculated as in Table 1.

Standard Error of Variance Component and Heritability

The general formulas for the standard error of a variance component are (Anderson and Bancroft, 1952)

$$Var(\sigma^2_g) \simeq \frac{2}{k_1^2} \sum_g \frac{MS^2_g}{df_g + 2}$$

$$S.E.\ (\sigma^2_g) = \sqrt{var(\sigma^2_g)}$$

where, k_1 = coefficient of the variance component

MS_g = g^{th} mean square used to estimate the variance component, and

df_g = the degrees of freedom of the g^{th} mean square.

An approximate method for the standard error or heritability is (Swiger et al., 1964)

$$S.E.(h^2) = 4\sqrt{\frac{2(N - 1)(1 - t)^2[1 + (k_2 - 1)t]^2}{k_2^2(N - f)(ft)}}$$

where, t = the intraclass correlation

N = total number of trees

k_2 = number of trees per each family, and

f = number of families.

Regression of Genetic Parameters Versus Infection Level

In this study, infection level implies the average percentage of trees infected in a test. Relationships between different infection levels and estimated values of heritability were explored. Change of the expression of genotypes through different environments was observed, since the same genotypes were replicated over three locations. Several different infection levels were interpreted as a series of different possible environments.

The efficiency of each test for detection of resistance to fusiform rust can be approximately determined from the average infection level of the test. The most efficient range of mean infection level can be determined through the expression of heritability and gain at that level of infection. Changes of the estimates of total phenotypic variance and additive variance were also projected over all infection levels. Several regression models were tested to find the optimum equation to define the relationships between the mean infection levels and these genetic parameters.

Breeding Methods

The heritability estimates computed from the above formulas were used in gain prediction. The equation for genetic gain or 'response to selection' can be more reliable if several assumptions can be met or accounted for in some way. These assumptions are

1. there is no contraselection on the side of the parasite,
2. there are no genotype x environment interactions, and
3. there is strong correlation of character expression in different stages of development.

Each assumption will be reviewed in the discussion section as it related to results of this study.

Expected gains from four different selection systems were examined for both types of populations. Realized gain was computed if possible.

The first system is mass selection in which all the rust-free trees or a random sample of rust-free trees were selected according to their phenotypes. The population-B itself is the typical example, where only rust-free parent trees are selected and used to produce the next generation. All seed production areas and seed tree stands belong to this kind of selection system. Predicted genetic gain from this system is

$$\Delta G_1 = i_1 \, K \, \frac{\sigma^2_A}{\sigma_1}$$

where, ΔG_1 = expected genetic gain from mass selection

i_1 = selection intensity based on the proportion of rust-free parent trees

K = fraction of the total additive variance in the covariance of additive value (e.g., K = 1/4 for half-sibs)

σ^2_A = additive variance

$\sigma_1 = \sqrt{\sigma^2_t + \sigma^2_{b(\ell)f} + \sigma^2_{\ell f} + \sigma^2_f}$ where

σ^2_t = total within plot variance which includes environmental variance of the small plot (σ^2_W) plus the remaining genetic variation depending on the family structure of the experiment ($3/4\ \sigma^2_A$ for half-sibs)

$\sigma^2_{b(\ell)f}$ = the error variance of large plots estimated as a family x replication interaction

$\sigma^2_{\ell f}$ = the genotype x environmental interaction estimated as a location x family interaction,

all other symbols are the same as defined previously.

The value of K for population-B is 1, since both parents are selected and contamination of pollen from other pine trees was prevented through isolation, whereas K can be half if only one parent is selected. In population-A, the mass selection involved selection of rust-free trees in a forest stand and the collection of their open-pollinated seeds. In this study the selection intensity was almost zero, since there was not enough rust when the mass selection was performed. Thus very little selection pressure was creased naturally. As a result, population-A is almost the same as the original population in terms of rust resistance and all the genetic parameters computed from population-A are applicable for computation of gains from selection systems. The gain from mass selection in population-A is assumed to be zero.

The second system of selection involved mass selection combined with progeny testing. In population-A, this procedure is represented by a rogued seed orchard of the best clones according to performance of their progenies, or establishing a new orchard including the best clones. In population-B, the seed production area is rogued to the best trees based on their progeny performance. Predicted genetic gain from this system is

$$\Delta G_2 = \Delta G_1 + 2i_2 \frac{\frac{1}{4}\sigma^2_{A'}}{\sigma_2}$$

where,

i_2 = selection intensity determined by the proportion of trees selected among those originally selected

$\sigma^2_{A'}$ = additive genetic variance of selected parent population $\sigma^2_{A'} = \sigma^2_A(1 - \beta v')$, where $\beta = h^2 i_1$ and is tabulated by Finney (1956). $\sigma^2_{A'}$ can be assumed same as σ_A in population-A, but not in population-B

σ^2_2 = variance of half-sib family mean

$$= \frac{\sigma^2_t}{k \cdot b \cdot \ell} + \frac{\sigma^2_{b(\ell)f}}{b \cdot \ell} + \frac{\sigma^2_{\ell f}}{\ell} + \frac{1}{4}\sigma^2_{A'}$$

The first two systems, mass selection and mass selection combined with progeny testing produce gains in the first generation of progenies from selected trees. The third and fourth selection systems are involved in selection among first generation progenies and the gains would be for the second generation.

The third type of selection system is to select the best individuals in the best families. These best individuals can be used for clonal seed orchards by grafting or the progeny test in which the best individuals were selected can be converted into a seed orchard. In this study, selection of best half-sib families from open-pollinated progeny tests and further selection of rust-free trees within half-sib families were carried out in both populations. However, in population-B, the expected gain from this selection system might be underestimated because both parents were already selected for resistance. Since the selection of the best families is based on general combining ability rather than specific combining ability, the same formula is applicable to population-B. The formula for gain from this selection system is

$$\Delta G_3 = \Delta G_1 + i_2 \frac{\frac{1}{4}\sigma^2_{A'}}{\sigma_2} + i_3 \frac{\frac{3}{4}\sigma^2_{A''}}{\sigma_3}$$

where,

i_3 = selection intensity determined by the proportion of individuals, uninfected within families that were saved

σ^2_3 = within half-sib family variance

$\sigma^2_{A''}$ = additive variance of the population after selection, $\sigma^2_{A''} = \sigma^2_A$, since genetic recombination has occurred.

The fourth type of selection is to select rust-free individuals in progeny tests for use as a seed production area. Alternatively open-pollinated seeds from plus trees may be bulked, the offspring planted at close spacing and

thinned later on the basis of individual phenotype only. The gain would be

$$\Delta G_4 = \Delta G_1 + i_4 \frac{\sigma^2_{A'''}}{\sigma_4}$$

where,

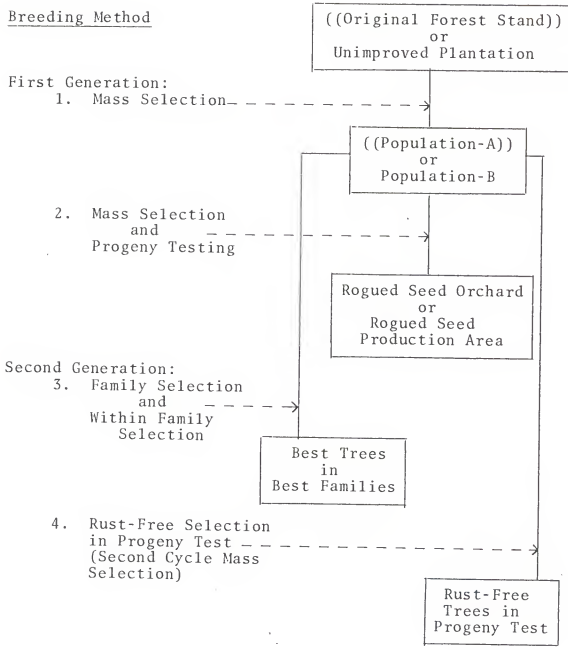
i_4 = combined intensity of i_2 and i_3, determined for rust resistance by proportion of trees uninfected

$\sigma^2_4 \simeq \sigma^2_3$

$\sigma^2_{A'''} = \frac{3}{4} \sigma^2_{A''} + \frac{1}{4} \sigma^2_{A'}$

A summary of these four selection systems in listed in Table 3.

Table 3. Summary of four different selection systems.



RESULTS AND DISCUSSION

Variation in Resistance to Fusiform Rust

The average infection level for progeny of population-A varied greatly among locations (Table 4). The 1971 Webster County, Georgia, planting had the highest average infection level, 85.6% in third year and 91.2% in the fifth year. There was much planting year variation in the Bulloch County, Georgia, site. There, 1971 planting averaged 51.1% infection in the third year, whereas the 1972 planting averaged only 13.1% infection. Infection in the other two locations remained very much the same over the two planting years.

Much variation in resistance to fusiform rust was also observed among families within a location. For example, rust susceptibility among families varied from 0% to 91% in the 1971 Taylor County planting.

The same trend in variation in resistance to fusiform rust was observed in population-B (Table 4) with less amount of rust infection than population-A in the same planting site. The standard deviation of family means for population-B was not any less than population-A in the same infection level category.

Effects of locations, blocks, families, and the interaction between blocks and families were statistically

Table 4. Range and standard deviations of family means, mean infection levels, and number of families included in statistical analyses of each location and planting.

	Year of planting	No. of families	Location	Age	Range	Mean	Standard deviation
				(yrs)	(%)	(%)	
Population-A	1971	92	Webster	3	41-100	85.6	12.67
				5	62-100	91.2	9.03
			Taylor	3	0-91	40.4	18.50
				5	9-100	51.5	18.73
			Bulloch	3	0-89	51.1	17.36
				5	11-93	57.2	17.42
	1972	64	Webster	3	25-96	61.7	16.02
			Taylor	3	12-83	46.2	16.46
				5	12-83	51.4	16.76
			Bulloch	3	0-44	13.1	9.46
				5	4-70	24.9	12.56
Population-B	1971	92	Webster	3	27-96	62.0	15.07
				5	31-100	70.3	15.20
	1972	92	Taylor	3	0-57	26.8	11.59
				5	3-61	29.7	12.35
			Bulloch	3	0-19	5.8	4.17
				5	0-28	12.4	7.23

Table 5. Analysis of variation in the amount of rust observed as a threshold character on the basis of individual tree values (binomial data).

Source	1971 planting		1972 planting	
	d.f.[a]	M.S.	d.f.[a]	M.S.
Locations	2	124.949**	2	101.776**
Blocks (locations)	6	1.158**	6	0.981**
Families	91	1.264**	63	1.114**
Locations x families	182	0.283*	126	0.247N.S.
Blocks (locations) x families	518	0.222**	369	0.230**
Within plots	6037	0.180	4561	0.186
Total	6836		5127	

** Significant at the 0.01 level

* Significant at the 0.05 level

N.S. Lack of difference at the 0.05 level

[a]Degrees of freedom of some sources of variation reduced due to missing data

Table 6. Analyses of variation in the amount of rust observed as percentage of infection per plot (plot mean).

Source	1971 planting		1972 planting	
	d.f.[a]	M.S.	d.f.[a]	M.S.
Locations	2	15.066**	2	11.723**
Blocks (locations)	6	0.120**	6	0.101**
Families	91	0.155**	63	0.122**
Locations x families	182	0.037*	126	0.027[N.S.]
Residual	520	0.029	368	0.026

** Significant at the 0.01 level

* Significant at the 0.05 level

N.S. Lack of difference at the 0.05 level

[a]Degrees of freedom of some sources of variation reduced due to missing data

Table 7. Analyses of variation in the amount of rust observed as percentage of infection per plot (transformed).

Source	1971 planting		1972 planting	
	d.f.[a]	M.S.	d.f.[a]	M.S.
Locations	2	66130.151**	2	50405.844**
Blocks (locations)	6	505.733**	6	442.934**
Families	91	657.572**	63	527.491**
Locations x families	182	145.089[N.S.]	126	112.769[N.S.]
Residual	514	120.844	363	113.367

** Significant at the 0.01 level

N.S. Lack of difference at the 0.05 level

[a]Degrees of freedom of some sources of variation reduced due to missing data

significant ($p < .01$) in all three analyses, binomial (Table 5), plot mean (Table 6), and transformed plot mean data (Table 7), and in both planting years.

The only variability in results of tests of statistical significance between analyses was found in tests of location x family interactions. Location x family interaction in the 1971 planting was significant ($p < .05$) with binomial (Table 5) and plot mean (Table 6) data but not with transformed plot mean data (Table 7). However, location x family interaction in the 1972 planting was not significant with any of the three analyses.

The F values for mean squares of location x family interaction of 1971 planting for binomial, plot mean, and transformed plot mean data were 1.272, 1.298, and 1.201, respectively. With degrees of freedom of 200 and 400, any F value larger than 1.22 is statistically significant at $p < .05$. With this many degrees of freedom even small effects can be tested with precision, though they may be of marginal practical importance.

Highly significant differences among blocks are not easy to explain because the plantings were arbitrarily divided into three closely adjacent blocks without regard to site conditions. In a sense, the large block effect is beneficial since some unknown source of variation was removed through blocking.

Different patterns of variability in two planting years have been reported by Blair (1970), in which male groups

were different statistically ($p < .01$) in the 1963 planting and not different in the 1964 planting. Rockwood and Goddard (1973) also reported the strong influence of year and site effects on rust susceptibility. Likewise, influence of sites were reported in several studies (Barker, 1973; Kraus, 1969; Wells and Switzer, 1971). It is interesting to note that in all years and locations in which representatives of both population-A and population-B were established, rust incidence in population-B was always substantially lower than population-A (Table 4).

Variation among slash pine families in rust susceptibility and resistance has been well established as one of the sources of resistance (Barber, 1964; Goddard and Arnold, 1966; Schmidt and Goddard, 1971).

Similar pattern of variation in population-B, selected population, as population-A agreed with some findings that variation in rust resistance among progenies of rust-free trees were significant even though they were much less infected than progenies from rust-infected trees (Jewell and Mallett, 1967; Dinus, 1971).

Estimates of Variance Components and Heritability

Estimates of the family variance component were very consistent in both plantings in all three types of analyses (Tables 8, 9, and 10). Standard errors for the family component were relatively small in all sites. Estimates of the

Table 8. Components of variance, standard errors of components and heritability estimates of resistance to fusiform rust observed as a threshold character on the basis of individual tree values (binomial).

	1971 Planting				1972 Planting			
	Webster	Taylor	Bulloch	Combined	Webster	Taylor	Bulloch	Combined
$\hat{\sigma}^2_f$ *	0.0120	0.0186	0.0157	0.0140	0.0142	0.0161	0.0034	0.0110
$S(\hat{\sigma}^2_f)$	0.0024	0.0047	0.0043	0.0025	0.0042	0.0050	0.0017	0.0025
$\hat{\sigma}^2_{\ell f}$				0.0024				0.0006
$S(\hat{\sigma}^2_{\ell f})$				0.0012				0.0012
$\hat{\sigma}^2_T$	0.1223	0.2412	0.2472	0.2011	0.2348	0.2487	0.1173	0.2028
$\hat{h}^2_i$	0.393	0.308	0.253	0.280	0.241	0.259	0.115	0.217
$S(\hat{h}^2_i)$	0.073	0.066	0.057	0.046	0.067	0.067	0.035	0.045
$\hat{h}^2_f$	0.752	0.604	0.570	0.787	0.613	0.589	0.389	0.782

*$\hat{\sigma}^2$ = estimate of component of variance, $S(\hat{\sigma}^2)$ = standard error of estimate of component of variance, f = family, ℓf = location x family, h^2_i = estimate of heritability of individual trees, and h^2_f = estimate of heritability of family means.

Table 9. Components of variance, standard errors of components and heritability estimate of resistance to fusiform rust observed as percentage of infection per plot (plot mean).

	1971 Planting				1972 Planting			
	Webster	Taylor	Bulloch	Combined	Webster	Taylor	Bulloch	Combined
$\hat{\sigma}^2_f$ *	0.0122	0.0198	0.016	0.0147	0.0138	0.0167	0.0032	0.0110
$S(\hat{\sigma}^2_f)$	0.0025	0.0046	0.0053	0.0027	0.0037	0.0046	0.0016	0.0025
$\hat{\sigma}^2_{\ell f}$				0.0028				0.0005
$S(\hat{\sigma}^2_{\ell f})$				0.0013				0.0011
$\hat{h}^2_f$	0.696	0.607	0.590	0.773	0.608	0.592	0.330	0.779

*$\hat{\sigma}^2$ = estimate of component of variance, $S(\hat{\sigma}^2)$ = standard of error of estimate of component of variance, f = family, ℓf = location x family, and h^2_f = estimate of heritability of family means.

Table 10. Components of variance, standard errors of components and heritability estimate of resistance to fusiform rust observed as percentage of infection per plot (transformed).

	1971 Planting				1972 Planting			
	Webster	Taylor	Bulloch	Combined	Webster	Taylor	Bulloch	Combined
$\hat{\sigma}^2_f$ [a]	58.30	77.08	59.43	62.84	57.19	71.26	13.81	45.75
$S(\hat{\sigma}^2_f)$	12.80	19.39	15.99	10.87	17.40	20.62	8.37	10.44
$\hat{\sigma}^2_{\ell f}$				8.07				N.C.[b]
$S(\hat{\sigma}^2_{\ell f})$				5.62				5.45
$\hat{h}^2_i$	0.265	0.327	0.258	0.294	0.253	0.285	0.066	0.206
$\hat{h}^2_f$	0.685	0.607	0.572	0.795	0.609	0.575	0.320	0.786

[a] $\hat{\sigma}^2$ = estimate of component of variance, $S(\hat{\sigma}^2)$ = standard error of estimate of component of variance, f = family, ℓf = location x family, h^2_i = estimate of heritability of individual trees, and h^2_f = estimate of heritability of family means.

[b] N.C. = negative estimate of component.

family variance component in binomial data and plot mean data were almost identical. The 1971 Taylor County planting showed the highest value for family variance. This also affected the estimate of individual tree heritability.

Estimates of family mean heritability for the combined locations were 0.78. These estimates were remarkably consistent among the three analyses.

The individual tree heritability estimates for the combined locations from binomial and transformed plot mean data were 0.280 and 0.294 for the 1971 planting and 0.217 and 0.206 for the 1972 planting.

Estimates of individual tree heritability varied greatly in binomial data from one location (0.115) to another (0.393) (Table 11). The only noticeable differences in the estimates of individual tree heritability between binomial and transformed plot mean data were found in the 1971 Webster County planting and in the 1972 Bulloch County planting. In both cases, the infection levels were very close to their extreme range (85.6% and 13.1%) and estimates from binomial data were much higher than estimates from transformed plot mean data. Otherwise, estimates from transformed plot mean data were higher than those from binomial data, but the differences were negligible.

The Robertson and Lerner method of analysis gave much higher estimates of heritability than other methods. With this method, a portion of the non-additive variance is included in the heritability estimate and this was particularly

Table 11. Comparison of estimates of heritability of individual tree computed from three different methods.

		Individual tree heritability		
		Binomial	Transformed Plot Mean	Robertson and Lerner Method
1971 Planting	Webster	0.393	0.265	0.462
	Taylor	0.308	0.327	0.352
	Bulloch	0.253	0.235	0.343
1972 Planting	Webster	0.241	0.253	0.271
	Taylor	0.259	0.285	0.341
	Bulloch	0.115	0.066	0.182

evident in the 1971 Bulloch County and 1972 Taylor County plantings (Table 11).

Considerable incluence on the magnitude of heritability estimates due to location difference has been demonstrated by Barker (1973). In most traits, estimates of heritability from combined locations were lower than any from separate locations. However, estimates for cronartium score, a severity scale, from combined locations were in between estimates from two locations, indicating additivity of estimates and no serious genotype x environmental interaction in rust resistance in loblolly pine.

In this study, estimates of individual tree heritability from combined locations were almost like mean values of the estimates from three locations separately. This agrees with the findings of Barker.

Differences in estimates of heritability between planting years were observed by Barker (1973) and Blair (1970). Differences in estimates between two planting years (0.217 vs. 0.280 or 0.206 vs. 0.294) in this study seem to be due to low infection level of one planting site in the 1972 plantings rather than to different patterns in variability of rust resistance between two planting years.

In general, estimates of heritability were somewhat higher than most of values reported by others, as seen in Table 12.

Table 12. Estimates of heritability of fusiform rust resistance reported in previous studies.

Authors	Tree Species	Year of Planting	Estimates of Heritability Individual	Family	Method of Analyses
Barker (1973)	loblolly	1960	0.11,0.35(0.23)[a]		Severity index
Blair (1970)	loblolly	1963	0.39,0.36(0.29)		NGT[c]
		1964	0.03,0.12(0.09)		NGT
		1963	0.25,0.21(0.22)		Severity index
		1964	0.02,0.09(0.04)		Severity index
		1963	(0.20)		Robertson & Lerner[d]
		1964	(0.12)		"
Goddard and Arnold (1966)	slash	1964[b]	0.199		"
Kinloch and Stonecypher (1969)	loblolly	1960		0.65-0.81	TPM[e]
		1960		0.72-0.85	NGT
Rockwood and Goddard (1973)	slash	1964-1967	0.052-0.238(0.054)	0.174-0.670(0.623)	TPM

[a]Where more than one value is shown, they are for different plantations and the combined heritability is shown in parentheses.

[b]Artificially inoculated.

[c]Number of galls per tree.

[d]Robertson and Lerner's (1949) method.

[e]Transformed plot mean data.

Heritability on Binomial Scale (p-Scale) Vs. Heritability on Normal Scale

Rust resistance was considered in this study as a threshold character which is discrete on the phenotypic scale and apparently continuous on the genetic scale. On the visible scale, individuals can have only two values, 0 or 1. Groups of individuals, however, such as families or the population as a whole can have any value, in the form of the proportion or percentage of individuals in one or the other class.

One of the shortcomings of heritability on p-scale is that the observed variance in p-scale data is correlated with the mean and hence becomes very small when the average percentage in either class approaches zero. This relationship makes the directly observed estimates of heritability depend in part upon the average incidence of the trait, unlike the situation in normally distributed characteristics. Heritability on the actually observed p-scale can be transformed into the genetically more accurate heritability on the normal scale by multiplying it by $\frac{p(1 - p)}{z^2}$ where p is the percentage incidence and z is the height of the ordinate which truncates p of the area of the normal curve (Dempster and Lerner, 1950). This technique has been extensively used in many papers to convert binomial heritability to normal heritability (Hill and Smith, 1977; Lush et al., 1948; Van Vleck, 1972). Estimates of heritability from binomial data in this study were converted into normal heritability with results as in Table 13.

Table 13. Comparison of heritability of resistance on p-scale and normal scale at varying incidence of fusiform rust.

Incidence (%)	Heritability on	
	p-scale (h^2_b)	normal scale (h^2_x)*
85.6	0.393	0.920
40.4	0.308	0.495
51.1	0.253	0.398
61.7	0.241	0.389
46.2	0.259	0.411
13.1	0.115	0.338

* $h^2_x = h^2_b \frac{p(1 - p)}{z^2}$

This probit transformation increased heritability on p-scale more than double when incidence was very low or high. Validity of transformed normal heritability is very questionable in this trait, rust resistance, especially in the test with high infection level.

Interpretation of normal heritability is a matter of open discussion but these values seem to be of very low practical importance. One possible interpretation can be made in relation to the performance of population-B which resulted from intensive selection for resistance. Of 92 families in population-B, only five in the 1971 Webster County planting were infected heavier than the average infection of population-A. The remaining 87 families can be considered as resistant if the average infection in

population-A is taken as the threshold point. The high normal scale heritability (.92) does not then appear inconsistent with the proportion (94%) of population-B families below the threshold point.

Assumptions for Accurate Gain Estimation

One of the assumptions, strong correlation of characters expressed in different stages of development, can be tested through simple correlation analyses. Usually as the tree gets older the resistance increases (Goggans, 1949). All of the rank correlation coefficients between third and fifth year rust data were highly significant (Table 14). Even the Bulloch County site, in which the third year infection was merely 13.1%, showed a rank correlation of 0.75 in population-A and 0.63 in the less-infected population-B.

Good correlations between artificial inoculation and field tests (Dinus, 1969; Wells and Dinus, 1974) also suggest that infection at the cotyledon stage could be well correlated with the performance of older trees. Some discrepancies between artificial inoculation and field tests (Goddard and Schmidt, 1971) may be due to the environmental factors rather than to actual differences in relative resistance.

A lack of genotype x environment interaction in resistance to fusiform rust has been confirmed in loblolly pine, particularly in regard to distinctive patterns of geographic

Table 14. Rank correlation coefficients (R) between third and fifth year fusiform rust data.

		Location	R
population-A	1971 plantings	Webster	0.77**
		Taylor	0.93**
		Bulloch	0.91**
	1972 plantings	Taylor	0.91**
		Bulloch	0.75**
population-B	1971 planting	Webster	0.92**
	1972 plantings	Taylor	0.96**
		Bulloch	0.63**

**Significant at the 0.01 level.

variation (Wells and Wakeley, 1966; Kraus, 1967; Blair, 1970; Wells and Switzer, 1975).

In contrast, some of the findings in slash pine were very disappointing -- no distinctive geographic pattern, and several indications of genotype x environment interaction (Gansel et al., 1971; Rockwood and Goddard, 1973; Sohn et al., 1975). Several geographic sources of resistance have been reported (Goddard and Arnold, 1966; Snyder et al., 1967; Sohn et al., 1975) but the locations were rather sporadic and showed no pattern of variation. Snyder et al. (1967) attributed these sporadic resistant geographic sources to family differences rather than seed source differences.

Differences in the behavior of these two closely related pine species might be explained through the evolutionary process. It is believed that the loblolly pine has had a much longer relationship with fusiform rust (Wahlenberg, 1960), and that slash pine is rather new to fusiform rust since large scale commercial planting started during the last two decades (Goddard and Wells, 1976). Coevolution between loblolly pine and fusiform rust is suggested in the central part of the rust gene pool and rust fungi in this high incidence area appear to have reduced virulence and caused reduced damage and mortality in the loblolly pine populations. Low susceptibility of loblolly pine populations at the extremes of the species range may be due to introgression of resistance genes from shortleaf pine. It is generally believed that coevolution of host and parasite under

exclusively natural selection leads to an equilibrium with specific genetic features on both sides.

On the other hand, coevolution between slash pine and fusiform rust is still in its early stage. Rust is constantly increasing and developing new strains to attack the slash pine population. It is a difficult task to predict where this coevolution between slash pine and fusiform rust would eventually lead. It is likely to follow the pattern of loblolly pine, since results of this study seem to indicate that genotype x environment interaction is only marginal.

If this is the case, bringing new resistant materials from other places to the highly infected area may confuse the course of natural selection and coevolutionary impact may not be as good as we expect. On the other hand, this evolutionary course might be led in a completely different direction in which the resistant material would eventually survive in the center of the rust gene pool. In this case, more slash pine families should be brought to these highly infected areas for ultimate sources for resistance. But the result from the most severely infected planting, the Webster County site, is rather disconcerting, since even resistant materials were infected up to 62% in the third year and 70% in the fifth year.

Potentiality of the threat of new strains of rust fungi has been well shown through the breeding work of cereal rusts, where new resistant cereal varieties had to be

developed every few years to meet the virulence of new strains of rust fungi (National Academy of Sciences, 1972). This may not be exactly applicable to pine rust breeding, since the pine population is rather heterogenous. On the whole, the pathogenic variability in different areas (Snow et al., 1969, 1972, 1975; Snow and Kais, 1970) seems to be very threatening, because this variability may be a future threat to the resistant materials selected presently.

Interaction between planting years and pine families was not tested in this study. Insufficient families were replicated in both years. Concerns about year x family interaction have been increasing. Blair (1970) found different patterns of inheritance between two successive planting years in loblolly pine. Rockwood and Goddard (1973) also found a significant difference in the interaction between year and progenies. These authors were unable to verify clearly the existence of this kind of genotype x environment interaction, since not enough materials were duplicated in each planting year and infection levels were not high.

An example of one family in the present study can illustrate the possible threat of year x family interaction.

		Deviations from Mean (%)			
Family	Year	Webster	Taylor	Bulloch	Rank
53-55	1971	-38	-35	-41	1 out of 92
	1972	6	1	5	37 out of 64

The performance of family 53-55 was the best in 1971 planting among 92 families but its rank dropped to 37th out of 64 families in 1972 planting. It is very unlikely that all other families included in 1972 planting were more resistant to fusiform rust than slash pine families included in 1971 planting. Their consistent performance in three different locations is very encouraging but such a big difference between two successive planting years is difficult to understand.

Implications of the possible existence of family x year interaction in a breeding program would be even harder to accept for foresters. Further investigation through proper experimental design is greatly needed to solve this very essential problem before any kind of definite selection procedure is developed.

The duration of this study is too short for any conclusions to be drawn concerning contraselection in the parasite. The impact of contraselection in fusiform rust is not immediate as potential fungal strains of high virulence require time to develop a large population. Additionally, resistant pines have not been established in large areas with susceptible pines excluded. This aspect is certainly a great concern in breeding strategy since Snow et al. (1975) found that aeciospores collected from galls from resistant trees could readily infect resistant pine seedlings. However, they also mentioned that the variability among galls in the same tree very often exceeded the variability among

galls from different locations. The potential danger of contraselection will remain a question until substantial numbers of resistant pines have been established.

Relationships of Genetic Parameters With the Mean Infection Level

One of the objectives of this study was to detect changes in genetic parameters over the infection range of tests. To cover a wider range of the infection levels, fifth year data of the 1971 Webster County and the 1972 Bulloch County plantings were also included in the regressions (Table 15). Each genetic parameter was expressed as a function of the infection level as based on the regression model which provided the best fitness statistically. In all cases, the R^2 was very high (over 74%) indicating that the regression lines fitted the data well (Table 16).

Binomial Data. Estimates of heritability for individual trees increased linearly with the function, $\hat{Y} = 0.0989 + 0.3272X$, as the infection level increased (Fig. 1). The same tendency was also true for estimates of family mean heritability. This continuous increase of heritability with higher infection levels is partly due to the change of the estimates of the total phenotypic variance and additive variance as the infection levels rise. Observed total phenotypic variance and additive variance are shown in Figure 2.

The regression line with the best fit for the total phenotypic variance was expressed as a parabolic function, $\hat{Y} = -0.0023 + 0.9995X - 0.9935X^2$. As the intercept, -0.0023,

Table 15. Genetic parameters and mean infection level for each test used for regression of these parameters to the infection level (proportion).

Planting year	Planting site	Age	X	Y					
			Infection level	Binomial data				Transformed plot mean data	
				σ^2_T	σ^2_A	h^2_i	h^2_f	h^2_i	h^2_f
1971	Webster	3	0.856	0.1223	0.0482	0.393	0.752	0.265	0.685
		5	0.912	0.0853	0.0280	0.334	0.704	0.177	0.614
	Taylor	3	0.404	0.2412	0.0741	0.308	0.604	0.327	0.607
	Bulloch	3	0.511	0.2472	0.0628	0.253	0.570	0.258	0.572
1972	Webster	3	0.617	0.2348	0.0568	0.241	0.613	0.253	0.609
	Taylor	3	0.462	0.2487	0.0644	0.259	0.589	0.285	0.525
	Bulloch	3	0.131	0.1173	0.0136	0.115	0.389	0.067	0.320
		5	0.249	0.1860	0.0271	0.159	0.474	0.145	0.474

Table 16. Analyses of variance for regression of variance components and heritabilities on infection level.

Source of Variation	Binomial data								Transformed plot mean data			
	σ^2_T		σ^2_A		h^2_i		h^2_f		h^2_i		h^2_f	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Regression	2	0.0202**	2	0.0010**	1	0.0308**	1	0.0615**	2	0.0207**	2	0.0402**
Deviation from Regression	5	0.0000	5	0.0001	6	0.0027	6	0.0062	5	0.0020	5	0.0016
R^2		0.999		0.855		0.743		0.909		0.805		0.909

**Significant at the 0.01 level.

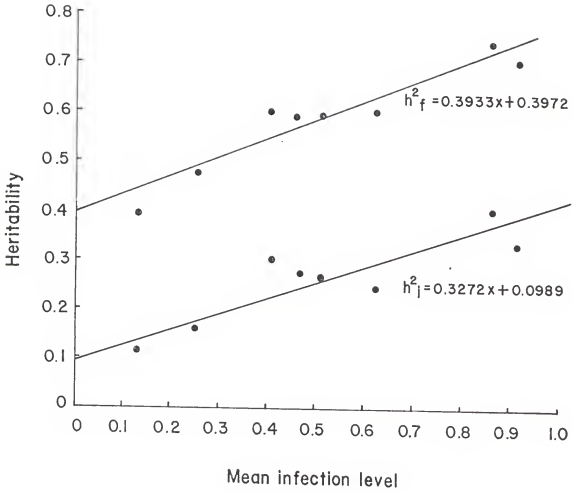


Fig. 1. Linear relationships between mean infection levels and the estimates of heritability of individual trees (h^2_i) and family means (h^2_f) computed from binomial data.

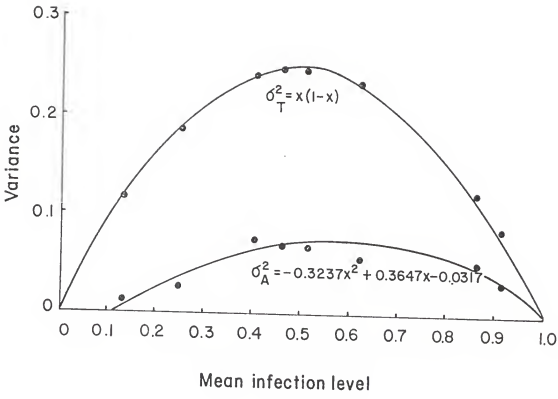


Fig. 2. Regressions of total phenotypic variance (σ^2_T) and additive variance (σ^2_A) of fusiform rust resistance computed from binomial scale data on mean infection levels.

is nearly zero, the function $Y = -X^2 + X$ can be stated $Y = X(1 - X)$ (Fig. 2), where X is the average infection level. In a binomial distribution function, the variance is pq, where p is the incidence and $q = 1 - p$. The average infection level, X, in binomial data is same as p, the incidence.

Estimates of the additive genetic variance increased up to the 40% infection level and stayed about the same to the 80% infection level, with a slight decrease beyond 80% (Fig. 2). The total phenotypic variance was very symmetrical with a peak at 50%. It decreased slowly after 50% and then sharply after 80%. Since heritability is the ratio of the additive variance over the total phenotypic variance and the denominator decreased sharply after 80% while the numerator decreased slowly, the increase of the estimates of heritability at high infection levels was inevitable.

Transformed Plot Mean Data. Estimates of heritability with transformed plot mean data showed a somewhat different relationship with infection level as indicated by the function, $\hat{Y} = -0.077 + 1.2418X - 1.0346X^2$ (Fig. 3). Since the additive variance changed very little through transformation and the sharp decrease of the total phenotypic variance for binomial data at high infection levels was moderated, the continuous rise of heritability over the infection level of 70% was not evident.

Further examination of the nature of the character of this trait, resistance to fusiform rust, and the estimates of heritability, led to a new function of heritability over

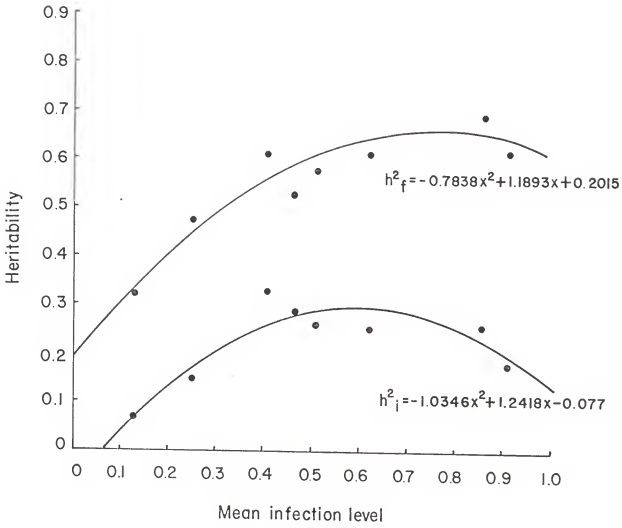


Fig. 3. Regressions of mean infection levels with the estimates of heritabilities of individual trees (h^2_i) and family means (h^2_f) computed from transformed plot mean data.

infection level. Logically, if the infection level is low, the chance for trees to 'escape' from infection is very high and the result of this 'escape' would cause a lower value of heritability. Once the test had enough inoculum to infect trees randomly and equally, then each family should be able to show the degree of resistance equally in each test, unless there is genotype x environment interaction. If the amount of inoculum was great or the virulence of the fungus was very high in a certain area, most families are completely infected and degrees of susceptibility cannot be expressed. The additivity of genes at higher disease incidence is only expressed among resistant families, whereas the additivity of genes was expressed only among susceptible families at low infection levels. In this case the estimates of heritability would be estimated to be lower than at optimum infection levels. A revised function for estimates of heritability over a range of infection levels is presented in Figure 4. The estimates of heritability increase linearly up to 40% infection level with a function, $\hat{Y} = 0.794X - 0.0366$. But the estimates of heritability remain constant between 40% and 85% levels of infection ($\hat{Y} = 0.281$) and sharply decrease at higher levels, likely expressed in a function, $\hat{Y} = -1.6744X + 1.7068$.

There is little literature available about the relationship between heritability in rust and infection level of the test. Rockwood and Goddard (1973) mentioned a positive correlation ($r = 0.40$) between heritability and infection level up to 70%. In most animal breeding studies (Dempster

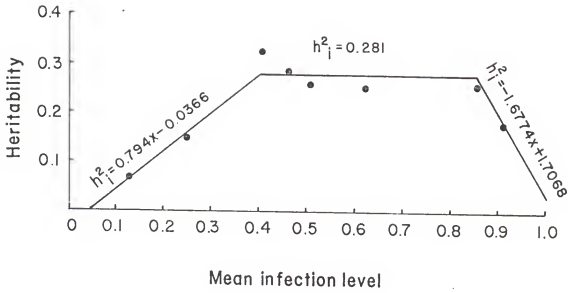


Fig. 4. Revised regressions between mean infection levels and the estimates of heritability of individual trees (h^2_i) computed from transformed plot mean data.

and Lerner, 1950; Van Vleck, 1972), however, symmetric estimates of heritability, peaking at 50% incidence, were assumed. This may well be true with random data in a statistical sense, but biologically, low heritability with low infection levels and higher heritability with higher infection levels seems to be a more plausible relationship. However, the high estimate of heritability with binomial data at very high infection levels may not be reliable, since environmental variance (total phenotypic variance for any given fixed genotype) of the underlying variate may not be independent of mean genotypic value (the level of incidence), as we observed.

Expected Gain from Mass Selection

Binomial Data. Expected gains from mass selection can be estimated with knowledge of mean infection level of the population where mass selection occurred (Table 17), since all three parameters which are essential for gain calculations were expressed as functions of the mean infection level. Among these parameters, selection intensity had the greatest effect on gains from mass selection. When the infection level is low, selection intensity is low as well as estimates of heritability and the total phenotypic variance of the population. A gain of less than 1 percentage point is expected with a low initial infection level (e.g., 10%). Higher gain is expected as the infection level rises. As mentioned, selection intensity in the most highly infected area (90%) is seven times larger than in the least infected

Table 17. Theoretical genetic gains from mass selection for different infection levels where mass selection is performed using binomial data.

Infection level (%)	σ^2_T [a]	i [b]	h^2_i [c]	Gain (%)	
				% point	% of mean
10	0.09	0.200	0.132	0.8	8.0
20	0.16	0.350	0.164	2.3	11.5
30	0.21	0.497	0.197	4.5	15.0
40	0.24	0.644	0.230	7.3	18.2
50	0.25	0.798	0.263	10.5	21.0
60	0.24	0.966	0.295	14.0	23.3
70	0.21	1.159	0.328	17.4	24.9
80	0.16	1.400	0.361	20.2	25.4
90	0.09	1.755	0.393	20.7	22.3

[a] Calculated from $Y = X(1 - X)$, where $Y = \sigma^2_T$ and X = infection level (proportion).

[b] Selection intensity tabulated with the proportion of selection, considered same as uninfected portion and infinite number of trees.

[c] Calculated from $Y = 0.3272X + 0.0989$, where Y = estimate of heritability based on individual trees (h^2_i) and X = infection level.

area (19%). The sharp decrease in the total phenotypic variance at higher infection levels did not interfere with high expected gain in binomial data, because of the seven fold difference in selection intensity in the highly infected population.

When the gain is expressed as a percentage of the mean infection level of the population, the differences in gain from different infection levels were rather small. The percentage point gain in resistance from mass selection was 20.7 in a 90% infected population, three times greater than the gain from mass selection from a 40% infected population, 7.3 percentage points. When the gain was expressed as a percentage of the mean of the population, however, the 22.3 percentage point gain from mass selection in the 90% infected population was not much different from the gain of 18.9 percentage points in a 40% infected population (Fig. 5).

Transformed Plot Mean Data. About the same amount of gain could be expected from mass selection using the transformed plot mean data (Table 18). Again, the selection intensity was the most important factor influencing the expected gain from mass selection. In the transformed plot mean data, the expected gain showed its peak at 80% infection and decreased thereafter. Utilizing two different values of heritability did not change the expected gain much in transformed plot mean data.

Variation of Gain from Mass Selection in the Next Generation. A more complicated problem lies in the

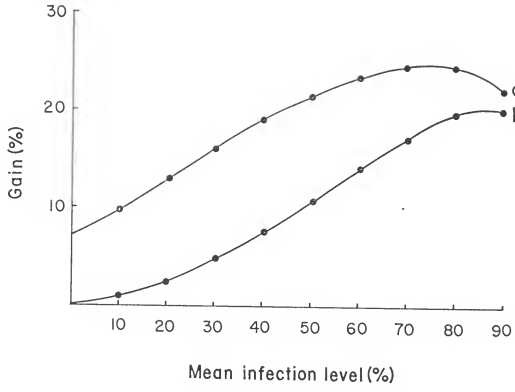


Fig. 5. Theoretical genetic gain from mass selection in p-scale data expressed as a percentage of the mean of the population (a) and as a percentage point (b).

Table 18. Theoretical genetic gains from mass selection for different infection levels where mass selection is performed using transformed plot mean data.

Infection level (%)	σ^2_T [a]	i	h^2_i [b]	Gain		h^2_i [c]	Gain	
				% point	% of mean		% point	% of mean
10	0.150	0.2	0.037	0.29	2.9	0.041	0.32	3.2
20	0.195	0.35	0.130	2.01	10.1	0.121	1.87	9.4
30	0.222	0.497	0.202	4.71	15.7	0.201	4.69	15.6
40	0.238	0.644	0.254	7.98	20.0	0.281	8.83	22.1
50	0.248	0.798	0.285	11.32	22.7	0.281	11.17	22.3
60	0.250	0.966	0.296	14.30	23.8	0.281	13.57	22.6
70	0.245	1.159	0.285	16.35	23.4	0.281	16.12	23
80	0.231	1.4	0.254	17.10	21.4	0.281	18.92	23.7
90	0.203	1.755	0.201	15.91	17.7	0.196	15.51	17.2

[a] Total phenotypic variance after transformation.

[b] Calculated from $Y = -1.0346 X^2 + 1.2418 X - 0.077$, where Y = estimate of heritability based on individual tree (h^2_i) and X = infection level (proportion).

[c] Calculated from revised equation for estimate of heritability

$Y = 0.794X - 0.0366$ when $X < 40$

$Y = 0.281$ when $40 \leq X \leq 85$

$Y = -1.6744x + 1.7068$ when $X > 85$

efficiency of mass selection of threshold characters. If the proportion of individuals required as parents is exactly equal to the incidence of the character in the population so that all such individuals and no others are used as parents in a mass selection program, the ensuing improvement would be exactly the same as that given in Tables 17 and 18. This is very possible in the case where the population was heavily infected and only a few would be left as parents for the next generation. When the population has a low infection level, and only half or less of the trees are needed as parent trees, the efficiency will drop and the theoretical gain from mass selection would be less than the maximum gain expected. Population-B was selected for rust resistance in a plantation where most of trees were heavily infected and all infected trees were removed and all of rust-free trees left for parent trees. Thus, the maximum gain from mass selection can be expected among progenies of population-B.

With binomial data, a gain of 20 percentage points, and with transformed plot mean data at least 16 percentage points, is expected in the case of population-B. The realized gains from mass selection were calculated from three different progeny tests, where the progenies of the population-A and population-B were randomly planted. In these tests, the realized percentage point gains from mass selection were 23.6, 20.0, and 7.2 for Webster, Taylor and Bulloch County, respectively (Table 19).

Table 19. Realized and expected percentage point gains from mass selection in progeny tests according to their mean infection level computed from two analyses methods, transformed plot mean and binomial data.

Location	Age	Infection level (%)	Realized gain (% point)	Expected gain (% point)	
				Transformed plot mean data	Binomial data
Webster (1971)	3	85.6	23.6	18.1	20.5
	5	91.2	21.1	11.3	17.3
Taylor (1972)	3	46.2	19.4	21.4	19.2
	5	51.4	21.7	21.6	20.5
Bulloch (1972)	3	13.1	7.3	4.1	7.4
	5	24.9	12.5	11.4	12.0

The concept of gain should be presented very carefully along with the nature of the test where the gain was estimated. The important factor is that gain from mass selection has been obtained in population-B when the infected trees were removed from the plantation. The three different values are just different expressions of gain from mass selection according to the condition of each test.

As indicated, the estimates of heritability and the total phenotypic variance varied along with the mean infection level of each test. In a way similar to that with which one predicts the gain from mass selection, the different expressions of gain can be predicted according to mean infection level of each test. The expected gain estimated in this way and the realized gain were very close (Table 19).

One puzzling fact was that expected gain computed from binomial data was much closer to realized gain, especially at the extreme ranges of incidences. Previously, we were concerned about the high risk involved in binomial data when the incidence is very high. This concern about estimates of gain from mass selection in high incidence can be moderated. At least in this study the gain from mass selection would be very precisely predicted with binomial data at the various infection levels.

The difference between estimates of the gain from each test was very apparent when gain was expressed as a percentage of the mean of the test. Gain expressed as percentage points (Fig. 6) showed a tendency to increase when the test site was

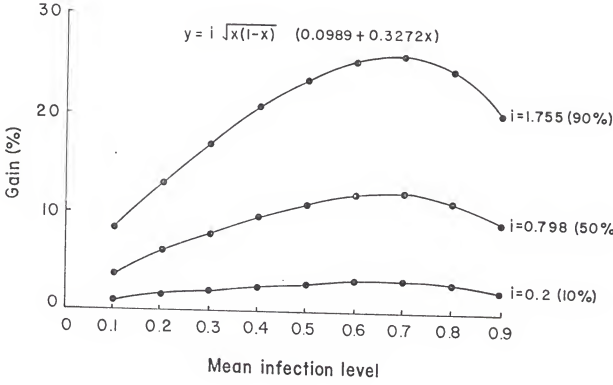


Fig. 6. Variation of expected gain from mass selection with mean infection levels in progeny tests, expressed as a percentage point. Different i values represent the difference in rust infection when mass selection was performed.

heavily infected, whereas the gain expressed as a percentage of the mean decreased almost linearly (Fig. 7).

As expected, the maximum unit gain from mass selection can be expressed when the test or the plantation has the optimum infection level. In this study the expected gain, rather an expression of the gain from mass selection for each infection, was a function of $Y = i(X(1 - X))^{\frac{1}{2}}(0.0989 + 0.3272X)$ as a unit (Fig. 6), and $Y = i \frac{1}{X} (X(1 - X))^{\frac{1}{2}}(0.0989 + 0.3272X)$ as a percentage of mean of the test (Fig. 7). The optimum infection level for the test to yield the maximum percentage point gain from mass selection was 71.0% according to the function. If the test material is less or more infected than 71.0% less gain would be expected in the particular test. Gain from mass selection also depends on selection intensity, which has a direct relationship with infection level of the population. The results from this study should be carefully compared with others, since each estimation was based on different situations. The nature of this study is closer to that of Dinus (1971) and the results agree very well.

Authors	Proportion Selected	Predicted (realized) Gain		Infection Tabulation
		Unit	% of Mean	
Barker (1973)	1%	1.4971	48.23	severity index
	50%	0.4481	14.44	severity index
Blair (1970)	11/37	8,4	16,9(25,2)	% infected
		0.71,0.07	24,3(17,0)	severity index
Dinus (1971)	Rust-free selection from plantation	(19.3)	(21)	% infected

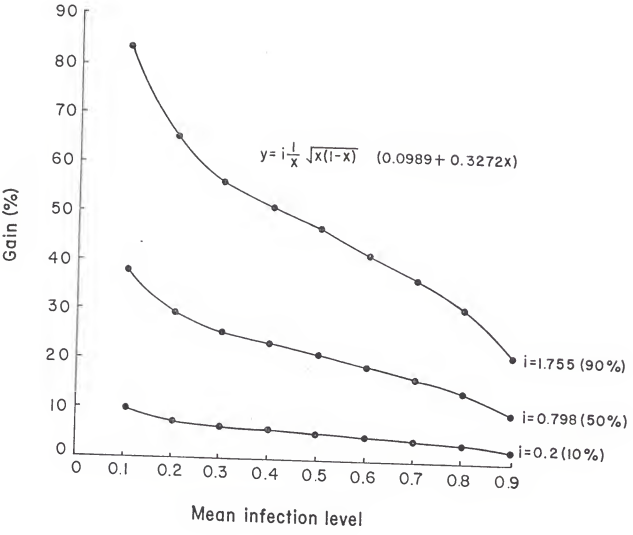


Fig. 7. Variation of expected gain from mass selection with mean infection levels in progeny tests, expressed as a percentage of mean. Different i values represent the difference in rust infection when mass selection was performed.

Expected Gain from Other Selection Systems

Mass Selection and Progeny Testing. This selection system is especially important for the current situation of most commercial seed orchards. Commercial seed orchards were established primarily with population-A type material which was selected mainly for growth and with very low selection pressure on resistance to rust. The easiest and fastest way to obtain the most gain would be te remove the susceptible clones from the seed orchard.

Approximately, gain of 23 percentage points can be expected when the best 25% of clones in a seed orchard are selected (Table 20). Gain expressed as a percentage of the mean varied according to the mean infection level of tests. In the 1972 Bulloch County planting, the gain was calculated from fifth year data which had an average infection level of 24.9%. The result was somewhat less percentage point gain (14.4%) than from other test sites, as expected. In general, realized gains were very close to expected gains.

In a test of higher heritability, like the 1971 Taylor County plantings, higher gain is expected than in other tests. Pathogenic variability in this area might be one possible reason for higher heritability at this test site. It is very important to plant the locally adapted clones when there is a possibility of genotype x environment

Table 20. Predictions of gain from three methods of selection in population-A when the best 25% of trees (families) were selected at age 3 years. Realized gains presented in the parentheses where possible.

		Predicted gain	
		% point	% of mean
Mass Selection and Progeny Testing			
1971	Webster	22.6	26.4
	Taylor	24.8	63.8
	Bulloch	23.2	45.4
1972	Webster	21.5	34.8
	Taylor	23.6	51.0
	Bulloch*	14.4	57.0
Family Selection and Within Family Selection			
1971	Webster	11.3(13.3) + 13.2 = 24.5	28.6
	Taylor	12.4(13.2) + 6.7 = 19.1	45.0
	Bulloch	11.6(12.3) + 6.2 = 17.8	34.8
1972	Webster	10.8(12.3) + 6.7 = 17.5	28.2
	Taylor	11.8(12.2) + 5.8 = 17.6	27.8
	Bulloch*	7.2(6.9) + 1.6 = 8.8	35.3
Rust-Free Selection from Progeny Test			
1971	Webster	22.2	25.9
	Taylor	10.2	25.2
	Bulloch	11.0	21.5
1972	Webster	11.7	18.9
	Taylor	10.1	21.6
	Bulloch*	3.4	13.6

*Calculation based on 5th year data due to low infection level in the 3rd year data.

interaction. So, the higher expected gain in this area might be applicable to this area only.

Additional gain of approximately 16 percentage points was predicted in population-B when the seed production area is rogued to the best 25% of the total trees in the area (Table 21). Gains from removing poor trees from the seed production area were smaller than those of population-A, since variation of the selected families was decreased through mass selection.

Selection intensity, the proportion of clones saved, affects the gain expected from this selection system considerably. To be very realistic, most commercial seed orchards can not afford to remove any more than half of trees from their original seed orchards. There is danger of inbreeding if the number of clones is reduced too severely, and other characteristics than rust resistance must be considered. On the other hand, establishing a seed orchard including only the best proven trees may result in much higher gain. One of the problems with this procedure is that most seed orchard managers would want to plant the same best trees and the potential danger of decreasing gene pools may be hard to avoid.

The necessity of progeny testing to increase gain has been emphasized in several studies, mainly due to poor parent-offspring correlation and great variability among progenies of rust-free trees (Blair, 1970; Dinus, 1971;

Table 21. Predictions of gain from three methods of selection in population-B when the best 25% of trees (families) were selected at age 3 years.

	Predicted gain	
	Unit	% of mean
Mass Selection and Progeny Testing**		
Webster (1971)	16.2	26.2
Taylor (1972)	16.8	62.7
Bulloch (1972)*	10.2	82.2
Family Selection and Within Family Selection		
Webster (1971)	8.1(14.7) + 9.3	28.1
Taylor (1972)	8.4(8.4) + 3.3	43.6
Bulloch (1972)*	5.1(3.6) + 0.7	46.8
Rust-Free Selection from Progeny Test (Second Cycle Mass Selection)		
Webster (1971)	16.2	26.1
Taylor (1972)	5.9	22.0
Bulloch (1972)*	1.6	12.9

*Calculation based on 5th year data due to low infection level in the 3rd year data.

**Initial gain from mass selection was not included (see Table 19).

Kinloch and Kelman, 1965; Jewell and Mallett, 1964, 1967). Dinus (1971) estimated 14% additional gain and Blair (1970) reported gain of 15% to 20% of the mean when mass selected trees were rogued based on progeny testing. Establishing a new seed orchard including the best 31% of the clones in the Bunswick Pulp and Paper Company seed orchard could ensure a gain of 20% over commercial check (Rockwood and Goddard, 1973).

Family Selection and Within Family Selection. In this study, calculations were based on selection among open-pollinated half-sib families and selection of the best tree from each selected family. One advantage of this selection system lies in the fact that each progeny test can be converted easily to a seed production area after the genetic information is gathered.

Selection intensity used for within family selection was based on the assumption that the best individual in each family would be chosen for use in a second generation seed orchard. However, selection intensity was determined on the basis of the average infection level of the best families selected. Each family was represented by a total of 30 or less individuals in one planting location and year. When the average infection level of selected families is 50%, within family selection implies random selection among 15 rust-free trees; the effective selection intensity

would be 0.777 even though only one tree is selected out of 30.

Approximately, gain of 18 percentage points can be expected from this system in population-A when the best individuals from the best 25% of the families are selected (Table 20). In the 1971 Webster County planting, the gain from this system was very high because of the large gain from within family selection. The same trend was found in population-B with highest percentage point gain from the 1971 Webster County planting (17.4%) and minimum percentage point gain from the 1972 Bulloch County planting (5.8%) (Table 21).

The ratio of gain from family selection and from within family selection varied greatly according to the mean infection level of each test. In a test with high infection level the gain from within family selection was almost same as the gain from family selection (1:1). In tests with moderate infection level the ratio between gains from family selection and from within family selection was 2:1. The ratio was completely reversed up to 5:1 in a test with very low infection when the most gain was due to family selection and within family selection did not contribute to the total gain. One of the advantages of this selection system is that the identity of selected trees can be preserved so that they can be used for a breeding population in advanced generations.

When 50 open-pollinated families were thinned to 20 and the best individuals (1/10) were selected within the best 20 families, a gain of 30% over commercial checks was estimated

(Rockwood and Goddard, 1973). Higher gains from family selection and within family selection from full-sib families were also reported by Blair (1970) and Rockwood and Goddard (1973).

Rust-Free Selection from Progeny Test (Second Cycle Mass Selection). This selection system can yield the most gain when the test is heavily infected. The expected gain from Webster County was as large as gains from the two previous selection systems (22.2 percentage points for population-A and 16.2 percentage points for population-B) (Tables 20 and 21). As the infection level decreased the gain decreased in the same manner as the gain from mass selection. In a sense, this selection system is almost the same as mass selection, and is described as a second cycle mass selection. Very little cost of management is one of the benefits of this selection system if the bulked seeds are used for establishment of the population in which the second cycle mass selection will be made. However, the identity of trees will be lost.

Rising concern over pathogenic variability in C. fusiforme along with the long rotation length of pines has to be considered. Establishing seed production areas in high rust incidence areas by this method -- planting bulked seed lots and later thinning to the best and rust-free trees -- seems to be an alternative to meet the continuous and abundant source of mutation of pathogen from existing virulent forms. Each location should establish its own progeny test to meet

local pathogenicity. A large number of families should be included in tests to maintain a broad gene pool, and a new progeny test needs to be established for every generation.

Selection for Wide Scale Resistance Vs. Selection for Resistance in Specific Locations

Alternative policies, regardless of specific breeding procedures adopted, are to develop a single, broad-based population for planting all sites or to develop a series of different populations, each particularly suited to a specific location. Where large genotype x environment interaction is found, the latter may be the most advantageous policy despite the added breeding costs involved.

There is also a potential danger from including families (clones) in a seed orchard which were not tested for the planting area. Potential gains in rust resistance from selection of the best families, over all three locations, in this study were compared with gains to be achieved by selection of the best families from each site separately (Table 22). In every case, planting of the best families selected in the same site had a definite advantage over selection based on all three locations. The greatest differences in these two procedures were evident in the 1971 Taylor County and the 1972 Bulloch County planting sites. In both cases, local selection for local use gave over 5% greater gain than selection of the best families over all three locations.

Table 22. Gains in fusiform rust resistance based on selection at age 3 years of the best 25% in individual or combined locations for planting in three locations tested.

Planted in	Selection based on			
	Combined locations (% point)	Webster (% point)	Taylor (% point)	Bulloch (% point)
Population-A				
1971 Webster	16.1	17.9	8.8	13.2
Taylor	16.7	12.5	22.0	13.1
Bulloch	19.3	17.7	12.3	22.0
1972 Webster	18.0	20.3	15.8	9.5
Taylor	19.6	17.6	21.3	11.3
Bulloch*	9.9	6.8	6.9	14.5
Population-B				
1971 Webster	16.9	21.1	6.7	2.3
1972 Taylor	9.8	6.0	14.1	0.2
Bulloch*	4.2	1.3	2.5	9.0

*Calculation based on 5th year data due to low infection level in the 3rd year data.

In all cases, selection on the basis of combined tests was superior to selection based on performance at a single location for planting at a different location. For instance, when selection was based on the 1971 Taylor County planting, performance of these families was much poorer in the 1971 Webster County and Bulloch County plantings than performance of selected families based on all three tests.

The magnitude of these differences was even more evident in population-B. It seems to be very important to include candidates of clones in a progeny test in the particular area where the planting is going to be done. From this, selection for locations where genotype x environment interaction is suspected should only be done in those locations and selection where infection level is very low is not effective for general resistance.

CONCLUSIONS

1. There was a substantial variation in resistance to fusiform rust among slash pine families even in a selected population (B) as well as in an unselected population (A).

2. There was an indication of family x location interaction, especially due to one test site, Taylor County, Florida. Year x family interaction was suspected even though the pattern of variation between two planting years was very similar.

3. Mass selection, phenotypic selection of rust-free trees in a heavily infected plantation, provided a very important initial gain for population-B (20 percentage points).

4. Combined estimates of heritability for individual trees and family means were very similar to two analysis methods: binomial data and transformed plot mean data. Expected gain from mass selection in binomial data was much closer to realized gain than that from transformed plot mean data. From this, it can be said that the expected gain can be estimated with confidence from binomial data.

5. The optimum infection level in progeny tests to obtain the most expected gain from mass selection was 71.0%.

6. Selecting the best 25% of the clones from a seed orchard or from a seed production area after progeny testing, or establishing a new seed orchard with best-proven trees will provide additional gains (approximately 23 percentage points for population-A and 16 percentage points for population-B).

7. Family selection and within family selection will also provide substantial gains for the second generation (approximately 18 and 12 percentage points for population-A and B).

8. Establishing seed orchards through recurrent rust-free selection in high rust hazard areas seems to be the best alternative to meet the challenge of pathogenic variability in C. fusiforme while maintaining moderate gain (approximately 10 and 6 percentage points for population-A and B).

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BIOGRAPHICAL SKETCH

Sung In Sohn was born February 2, 1948, in Seoul, Korea. In January, 1966, she graduated from Kyung-Ki Girls High School, in Seoul. In March, 1966, she entered the Seoul National University and received the degree of Bachelor of Science in Forestry, 1970. In September, 1971, she enrolled in the University of Florida and received the degree of Master of Science in Forestry in June, 1973. She has been working as a graduate assistant in the School of Forest Resources and Conservation.

She is a member of the Society of Suwon Foresters and the Society of Korean Foresters.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Ray E. Goddard

Ray E. Goddard, Chairman
Professor of Agronomy

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Charles J. Wilcox

Charles J. Wilcox
Professor of Dairy Science

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Earl S. Horner

Earl S. Horner
Professor of Agronomy

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Robert A. Schmidt
Associate Professor of Forestry

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Charles A. Hollis
Associate Professor of Forestry

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Anthony E. Squillace

This dissertation was submitted to the Graduate Faculty of the College of Agriculture and to the Graduate Council, and was accepted as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

August, 1977

Jack L. Fry
Dean, College of Agriculture

Dean, Graduate School